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ENTERALGIA

AND

CHRONIC PERITONITIS.

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Enteralgia means always an excessive irritation of a branch, or branches, of the sympathetic nerve. It is a better and more intelligent term than that of "colic;" and by its nature and seat, it is diagnosticated more or less readily by its differences both from the pain occasioned by renal or biliary calculi, and from a number of affections of the surface. Amongst the latter, I refer mainly to rheumatism of the abdominal muscles, hæmatoma of the same (mostly of the rectus) brought on either by over-exertion or purpuric changes in the blood-vessels; lumbo-abdominal neuralgia; or the neurotic sensitiveness of cutaneous nerves in hysterical persons.

Enteralgia being an affection of some nerve branch or branches of the intestine, its *cause* must be sought for either in the nerve itself and alone, or in a change of either the intestinal tissues, or the contents. The nerve may be affected directly by a hysterical and hypochondriac condition, by malaria and gout, and by poisons, such as lead; or the pain may be the peripherous result of a disease of the spinal cord; or it may be the reflected effect of an irritating affection of the liver or genito-urinary organs or the skin. The latter cause is quite frequent. Indeed, sudden refrigeration of the surface, "cold," is a more frequent occurrence than is claimed by some of those who look upon everything as obsolete and fallacious, only because it is old and has once been generally accepted.

The *anatomical changes* of the tissue resulting in enteralgia may vary between a simple congestion or nutritive disorder, and an inflammation with its results. Thus the congestion of the colon, connected with the prevalence of extensive rectal varicosities, gave rise to the term hemorrhoidal colic; and enteralgia depending on every form of enteritis and colitis, ulcerations (more frequently dysenteric than typhoid) and stenosis, is quite frequent.

Alterations in the contents need not be very great in some persons to give rise to enteralgia. Some are easily affected by certain articles of food, particularly acid ones. Certain drastics, such as senna, and many poisonous substances give rise to severe griping pain. The presence of hard scybala may so obstruct the bowels as to produce enteralgia by direct pressure or locking up gas. Putrid food, or fermentation of otherwise normal food, meeting insufficient or faulty digestive juices will have the same result. So will foreign bodies, both those that are swallowed, or such as are more permanent tenants, viz.: animal parasites.

The *definition and the exact nature of enteralgia* are not modified by the duration of the pain. It may be quite short or extend over a long time, and begin and end gradually or suddenly (paroxysmally), with temperature normal or subnormal, rarely elevated; pulse sometimes slow, sometimes innumerable; skin cold and clammy; sometimes dysuria, nausea and vomiting, constipation or diarrhœa. At the same time, the abdomen is tumid —either generally or locally; when the latter, the local tumidity is apt to change its place under inspection or on palpation. In some cases, there is no such inflation, but rather a retraction. This is so mostly in cases of strictly nervous origin; in others there is a spasmodic contraction of abdominal muscles and cremaster. With the spastic rise of the testicles, there may be connected priapism and seminal discharges.

It is the object of these remarks to direct your attention to one of the causes of enteralgia which is quite common, though it be very liable to be overlooked. But it is well to be aware always that pains may be different things; and when so, have different indications and require different treatment. Indeed, many of the attacks of pain called colic and enteralgia are only the symptoms of, mostly chronic, peritonitis.

Primary peritonitis of any kind is very rare; and then mostly traumatic, the result of wounds, probing and paracentesis for ascites included. Many more result from exposure—"cold."

Most cases are of a *secondary nature*, with very numerous causes. It may arise under the influence of general diseases—rheumatism, alcoholism, scarlatina, measles, erysipelas, malaria, scurvy, tuberculosis, and carcinosis. The inflammation of the neighboring thoracic cavity may be transmitted through the patent stomata of the lymph ducts on the upper and lower surface of the diaphragm; and disorders of circulation depending on pulmonary and cardiac diseases will result in congestion of the peritoneum, both visceral and parietal, with its possible consequences—viz., inflammation, or ascites, or both.

The presence of new formations in the abdominal cavity acts like that of a foreign body. Adhesions to an unexpected extent are often found during ovariotomies. A sarcoma of the kidney is often glued to the colon in front of it, or to other intestines; swelled pelvic glands, in connection with irritated inguinal and tumefied mesenteric glands, will often be found with secondary peritonitis. A young woman, whom I saw dying with acute peritonitis, had been affected with purpura for some weeks; at the autopsy, that peritonitis was found to be the consequence of haemorrhages from some ruptured vessels of the diaphragm.

Floating kidney, by its changing position and irritating contact with the peritoneum, is quite liable to light up a chronic inflammatory process, thus becoming fixed by adhesions, and dislocated after having been floating.

Affections of the vertebral column and adjoining organs will produce peritonitis; so will spondylitis, mostly tubercular, and abscesses of the bone; also thoracic abscesses following the course of the fascia downwards; and abscesses of the psoas and iliac muscles, or perforating abscesses of the socket of the hip-joint.

Amongst the most frequent causes of peritonitis are catarrhal and inflammatory diseases of the female sexual organs. Beside the opportunities afforded by menstruation—viz., salpingitis, parametritis, perimetritis, and endometritis—there is no more frequent mischief than that originating in the sexual functions of woman. Cohabitation is sometimes, the puerperal state very often, the cause of persistent peritonitis.

The irritation or inflammation of any of the organs contained in the abdominal cavity are liable to produce peritonitis. A swelling of the liver, from a simple secondary congestion to an abscess, or a hydatid cyst, which expands the covering of the organ, makes peri-hepatitis. The presence of a biliary calculus obstructs the choledoch duct, not so often by its presence as by the local peritonitis brought on by pressure. Perinephritis will often spread and lead to intra-peritoneal inflammation; diseases of the spleen and pancreas have the same result. The intussusception of the nursling leads always, on the locality of the invagination, to local hæmorrhage and inflammation; irritation and inflammation are quite frequent in the left hypogastrium of the young, where the normally long colon of the infant is sometimes folded in a number of flexures compressing each other; and about the right and the left curvatures of the colon, as the result of obstinate constipation, with dilatation and thickening of the colon.

I remember well the case of a woman who, while carrying twins, suffered at the same time from obstinate constipation. Her confinement was normal, and she left her bed in due time. But she would always afterward complain of a constant—sometimes dragging, sometimes acute—pain far away in her abdomen. Careful repeated examinations localized the pain in front of the spine, following the exact course of the abdominal aorta. The diagnosis of aortic perivasculitis could safely be made and was borne out by the further course of the symptoms. While she was getting much better, a trip in a jolting wagon over a mountain road gave her not only an increase of pain, but an extension of the local affection into a peritonitis, the consequence of which she will feel all her life-time.

The most frequent cause of peritonitis is, perhaps, a preceding peritonitis. Indeed, when a case is examined after death, the positive proofs are found of one or more attacks preceding the fatal one. Thus if not the proximate cause of death, at all events the main cause may be set down to have been a previous attack. I do not remember a case of perityphlitis but what exhibited the adhesions, discolorations and contractions due to former peritonitis; frequently the vermiform process was attached to the side or the posterior wall of the colon; the tissues of the intestine were thickened, the parietal peritoneum whitish and thickened, and the orifice of the process patent. Indeed, it is probable that there are few, if any, cases of foreign bodies entering the process unless the

latter have previously lost its elasticity and contractility by an inflammatory change.

Anatomical changes—Alterations of the mucous membrane of the intestine are the initial stages of local peritonitis in many instances; of general peritonitis in some. It is not only the intima and submucous tissues which are suffering, but the muscular layer is implicated in the morbid process. It is not necessary here to recall the histological changes; it suffices to point to the clinical and anatomical fact that a simple intestinal catarrh is growing easily and speedily into an enteritis. The vascular connection between the three principal layers of the walls of the intestine is such as to facilitate the transmission of an inflammatory process from one to another. Thus it is that a peritonitis —that is, the inflammation of the serous membrane—is communicated to the muscular andmucous tissues, thereby spreading œdematous infiltration, paralysis and constipation; thus also it is that a common diarrhœa is able to develop in a shorter or longer time a local peritonitis. If it were necessary to exemplify this intimate connection of the two, it would perhaps be a desirable illustration to recall the facility with which anatomical changes take place in the mesenteric glands in the course of a common diarrhœa. No morbid process can be expected to be isolated in a locality which is supplied with an active blood and lymph circulation. Thus it is that an intestinal catarrh grows to be an enteritis, the enteritis a peritonitis.

This condition of things is still more frequently observed in cases of intestinal ulceration, both acute and chronic. The most intense form is the peritonitis following the perforation of a gastric or intestinal ulcer with its speedy fatal termination. But without perforation, an ulceration will lead to peritonitis which is mostly local, or local in the beginning, and liable to change into an acute attack under favorable circumstances. It is very easy to verify the following condition of things: Where there is an open ulcer, or one that had cicatrized months or years before, in the stomach, or in the intestine, no matter of what nature, either catarrhal, or dysenteric, or typhoid, or tubercular, we frequently find opposite the ulceration or cicatrix that is in the peripheral covering, a local peritonitis. There is a thickening, circumscribed and distinct. In recent cases, it is rather soft and suc-

culent, with much vascular injection; in old cases, the orginal cell proliferation has undergone organization and hardening, the thickened spot is gray, whitish, hard, and has lost its elastic and soft feel. Not only does it *feel* less elastic; it *is* so. It is very apt to burst under a moderate amount of pressure, and lead to perforation. In the midst of apparent health, intestinal perforation will set in, and death ensue within a day. The mysterious catastrophe is explained by the autopsy which reveals a perforation in the midst of such a local peritonitis as I have described. Then only it is that the medical man will learn that the man called away so suddenly was the victim of the perforation of the cicatrix of a typhoid ulceration contracted a dozen years previously.

Many years ago, I presented to the Pathological Society of New York the specimens of the intestine of a child who died with intussusception not reduced. It was a very unusual case. The invaginated mass from six to eight inches long reached into the rectum. Many attempts at reduction were made—from the use of big sounds, to inflation of air and injection of water. At the autopsy half a dozen perforations were found, some of which were complete, some partial, in such a way that only the adventitia had bursted. None of the perforations were within reach of the instruments used for the purpose of relief; but around all the rents there was the peritonitis, thickening and discoloration, and the general anatomical changes described before; and generally the wall of the perforation was found raised beyond the level of the neighborhood, so that where the wall was thickest it was most fragile.

What was the cause of all this, in the apparently healthy and well-developed child? On inquiry only it was found that two years before the fatal attack the boy had suffered from "summer complaint" for a number of months; and that he had often had "cramps," crying spells, colics and the like, since.

The *diagnosis of chronic peritonitis* is frequently missed. As stated before, unforeseen adhesions are often found round tumors; moveable kidneys become fixed, intestines glued together—all without recognizable symptoms.

In chronic peritonitis, *respiration* need not be accelerated; particularly is that so in pelvic peritonitis, perimetritis, and pericys-

titis. There may be occasional *vomiting*, particularly where there happens to be an intervening acute catarrh; but there are other conditions—for instance, renal and biliary colic—which are more liable to exhibit that very symptom to an excessive degree. Indeed, it is so often absent in chronic peritonitis that its very absence, being a negative symptom, is not of much account. Even in many acute cases it is not met with. There is, for instance, none in twenty-five cases out of a hundred of septic peritonitis of the newly born. *Constipation* is frequent, diarrhœa not unusual. *Horizontal posture* is often quite uncomfortable; but a common colic depending on gas not absorbed or expelled in duces the same posture (viz.: the knees drawn up) in most instances. However, when horizontal posture is shunned in chronic peritonitis, the patient is more apt to be quiet with raised knees than he who is suffering from flatulency; very few of this class will abstain from kicking and moving. The *abdomen is apt to be tumid*, but it must not be overlooked that general adiposity is mostly developed on the abdomen; that women who have had children are liable to have large and prominent abdomens; that that of the healthy baby is so large as to measure one-third of his whole length; that a simple hysterical dilatation and inflation may simulate that which is produced by peritonitis; that with the former, there is sometimes an œdematous swelling of both hypogastric regions, which complicates the diagnosis still more seriously, and that there may occur a local dilatation of an intestine from habitual constipation only. The *surface of the abdomen exhibits nets of dilated veins* more frequently in peritonitis (and hepatic diseases) than in any other morbid condition. Now and then there is a friction sound or a slight crepitus *on auscultation; inspection* may also reveal solitary convolutions which rise above the level; and *palpation* may lead to the discovery of exudations in various shapes and sizes, nodules, lumps, cakes, hard and soft, which are either organized material or glued intestines. *Percussion* may discover these solid masses, or fluid contents. *Fluctuation* will show ascites more readily than percussion, for the latter may fail in this, as there may be adhesions between the parietal peritoneum and intestine in the flanks. The gas contained in the adherent bowel will then yield the tympanitic percussion note, though the region may be filled with fluid: a

change of posture, from side to side, or from the horizontal to the vertical, may contribute to dispel the doubt.

A chronic peritonitis is sometimes diagnosticated with more or less certainty in the following manner: The patient is on his back, extremities now extended, then again flexed. Pressure is tried—soft, hard, sudden, or gradual, superficial or deep, in the usual way. Often the seat of the *pain*, inflammation or adhesion is thereby made manifest. In many cases, however, the following manipulation answers best: Make deep pressure with the palm, fist, or a finger. Perhaps there is no pain. Relieve the pressure at once, and a local, very distinct and circumscribed pain may be felt. Repetition of the experiment will give the same result always, the symptom being elicited by the sudden change in the relative position of the bowels. Not only pain, but the presence of floating exudations can be distinguished by this and similar manœuvres.

Every change in the relative position of the bowels may rouse a pain. A sharp pain after a full meal may point to adhesions of the stomach; three or four hours after eating to chronic colitis; a quickened inspiration or a cough, to perihepatitis; toward the end of micturition, to pericystitis.

Pain is a very frequent symptom in chronic peritonitis. It may be mild, severe, of short duration, or persistent. Its variability depends often on the degree of irritation or congestion. Acute attacks are frequent when there is a cause for exacerbation.

On the 22nd of September, 1884, I saw a boy of eight years with Dr. G. He was known for years to have suffered a good deal from abdominal pain—sometimes for a short time, sometimes half a day. It was mostly located in the right hypogastrium; now and then in the epigastrium, and frequently complicated with constipation. It had disturbed him sometimes during school hours, and more so his elderly school-mistress who hated stomach ache occurring at improper times. About the 8th of September the child swallowed a cent—no serious consequences following. On the 22nd he went to school in perfect health, was taken with severe pain in the right hypochondrium at ten o'clock, was kept in by his intelligent teacher until noon, and, stooping, crept more than he walked until he finally reached the bed, from which he did not rise. The pain was intense, mostly on the right side; the abdomen was swollen correspondingly with the pain; the thighs were drawn up, temperature rose, and peri-

tonitis became general. On the 25th the temperature of the rectum decreased very much, and the child died in general collapse.

At the autopsy, which was made on the 26th, there were five hundred grammes of pus in the abdominal cavity; strong adhesions—old and new—of the intestines amongst themselves and with the abdominal wall; a poppy stone in a hard fecal mass in the vermiform process; one perforation, two gangrenous spots and firm angular adhesion of the vermiform process to the colon, of various thickness and shades of color, thus proving, and also by its hardness, the cause of the repeated attacks of local peritonitis the child had been suffering from for years, and explaining the many enteralgias complained of from time to time.

The seat of the pain varies with the location and the extent of the lesion. Extensive peritonitis in the pelvis may not give rise to pain except such as is waked up by defecation, cohabitation, or micturition. Attending pericystitis is well characterized by its pain, which appears when the urine has been voided about one-half and the bladder contracts more efficiently. In this it resembles much the spasmodic pain of vesical catarrh, with this exception, that it is more localized above the pubes, and manifests itself by pressure more readily.

In most cases, the pain of chronic peritonitis can not always be *diagnosticated from the enteralgia produced by other causes*, such as abnormal contents, fermentation, flatulency. Indeed, the anatomical changes of chronic peritonitis give rise to those other conditions. By it the intestinal movements are retarded; stenosis may result from it, also twisting and adhesions; and through them every function is seriously interfered with. If it were easy, or in many cases possible, to make the diagnosis of the pain, the mistakes would not be so frequent; and the condition of things of which you have permitted me to speak before you, could not to-day be the subject of our discussion.

The *results of chronic peritonitis* are very various. A simple attack of acute exudation may shape the future of the patient. I knew for years an elderly lady of perfect health, not disturbed except by flatulency, constipation and frequent enteralgia, which she dated back to one of her confinements. The diagnosis of dilated transverse and descending colon could be made easily. Purgatives administered in regular intervals and two daily enemata would keep her regular and without pain. The amount of water admitted to the bowels was large. Thus if there were a stenosis,

it must be at a distance from the rectum; besides, no vaginal or rectal examination revealed the presence of a tumor or swelling. When the autopsy was made many years afterwards, in Zurich, Switzerland, there were found many discolorations and thickenings of the adventitia of the colon, some old adhesions of the descending colon to the abdominal wall, and a hard and massive band extending from the left ovary to the junction between the middle and lower third of the colon; the latter was constricted to half its original size.

There was a girl of seven years in Bellevue Hospital five years ago. She entered with a history of enteralgia, emaciation, loss of appetite. Her pain lasted sometimes for hours, sometimes for days; followed exertion, exercise, eating; was sometimes mild, often excessive; raising the knees, passing of flatus, of urine, or belching would now and then relieve her—at other times not at all. Thus it appeared that the attacks were not always alike, and were probably the result of a variety of causes, partly mechanical, partly functional. The underlying cause of them all was chronic peritonitis. There was pain on pressure, some ascites, and a large number of swellings, more or less hard, of the size of a hazelnut or hen's egg, numerous, and big enough to be mistaken for neoplasms; now and then an exacerbation of temperature. All this made the diagnosis of chronic peritonitis safe enough. She was kept in bed for months, with careful diet and wine of pepsin; absolute rest, poultices, a daily enema, and iodide of potassium in the beginning, iodide of iron afterwards. She left the hospital well and hearty, with no perceptible tumors, with no pain for many weeks, and supplied with a bandage to protect her. She remained well, and died of a non-tuberculous pneumonia two years afterwards.

I was fortunate enough to obtain the *post-mortem* examination. There were whitish discolorations of the adventitia, and thickening of the intestinal wall in many places; very strong and numerous adhesions between the convolutions; thickening, and in part shriveling of the omentum, and a few hyperplastic glands of non-tuberculous character.

This was a classical case, but one of the unusually severe ones of the class the picture of which I have tried to draw for my fellow practitioners who are constantly called upon to meet them.

The histories of previous acute attacks of peritonitis are often not remembered. One of the greatest physicians and surgeons New York ever called its own, died fourteen years ago, during his convalescence from typhoid fever, of peritonitis. He had often complained of enteralgia and occasional attacks of diarrhœa,

during the twenty years I knew him intimately. But it was only during the first leisure of his life viz: the long weeks of his dying, that he remembered having suffered from peritonitis thirty years previously. At the autopsy we found discolorations in many places of the serous membrane; degeneration, dilatation, and atrophy of the colon to such an extent as to result in a number of perforations. From such a case it becomes evident that there are, and how it happens that there are, so very many extensive adhesions and other changes without any history whatsoever.

Treatment of Enteralgia Depending on Chronic Peritonitis.—The indications for treatment are given by its results and symptoms, amongst which are prominent, besides the pain, sluggishness of a part of the intestine, constipation, adhesions and bands, and intervening sub-acute and acute peritonitis. The latter require appropriate treatment, such as absolute rest, with support for the knees; ice or warm or hot applications according to circumstances, and opiates in sufficient doses. As a general treatment, the latter are more justified than the sulphate of magnesium and turpentine enemata recommended by some. For the treatment under which an occasional patient may escape death, must not supersede one which has proven to be successful in most cases, and beneficial in every one.

The localized attacks, mainly in the right hypochondrium, demand local applications; occasionally a few leeches, and subcutaneous injections of morphia may become necessary. Old adhesions and organized bands are not amenable to medicinal treatment; and the surgical interference sometimes demanded by such anatomical changes do not concern us here. A person afflicted with chronic peritonitis, such as I have described it, must not choose work which requires great physical exertion, straining and lifting; must avoid injuries, pressure on the abdomen, jumping, jolting, and straining during defecation, or working at the sewing machine, etc. His bowels must be kept regular by a daily enema, even when there is an occasional apparent diarrhœa, for this diarrhœa is often complicated with constipation and alternating with it.

Beside the daily enema, my main reliance is on the wearing of a snug bandage which must cover the whole abdomen, and is fastened low down by soft straps passing under the perineum,

both in front and behind. Thus, jolting and moving of the intra-abdominal contents are avoided. That bandage must be worn until the patients have not complained for years. In hundreds of cases I have known it to give immediate relief; without the immobility given by it to the sore intestine, I do not expect a case to do well.